

ABSTRACT OF THE DISCLOSURE

Anterior ischemic optic neuropathy (~~AION~~) is one
of a family of an ischemic diseases affecting the optic
nerve. A blockage of vessels supplying the intra-retinal
5 portion of the optic nerve causes results in loss of axon
transport stasis, retinal ganglion cell, (~~RGC~~)—specific
dysfunction, and RGC death. ~~AION~~ Research has been limited
by the lack of an appropriate, easily replicable, rapidly
inducible, low-cost models for this disease. We have
10 developed such a model. Animals were handled and utilized
in accordance with ARVO guidelines. Using a custom
designed fundus contact lens, an intravenous injection of
photosensitizing agent was administered to anesthetized
100g male Sprague-Dawley rats. A laser was used to
15 directly activated dye within the small vessels perfusing
the optic nerve. This treatment was used to selectively
spared the larger caliber vessels perfusing the inner
retina. Electrophysiologically, a decrease in amplitude of
the visual evoked potential is noted. Gross, histologic,
20 molecular, and electrophysiological techniques are used to
analyze changes induced by this method. The acutely
treated rodent optic nerve grossly has the appearance of
human AION, with pale edema. Electrophysiological, a
decrease in amplitude of the visual evoked potential is
25 noted. Histologically, alterations in axonal transport are
seen. Reverse-transcriptase based Polymerase chain

later retinal gene expression changes in the treated animals. This new method accurately replicates many cellular and molecular level changes in a low-cost animal model. ~~may greatly accelerate our understanding of the~~
5 ~~pathological long and short term processes involved in~~
~~AION, and increase our ability to develop more effective~~
~~treatments for this disease.~~